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Review Article

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# Recent Updates on Controversies in Decompressive Craniectomy and Cranioplasty: Physiological Effect, Indication, Complication, and Management

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## ABSTRACT

Decompressive craniectomy (DCE) and cranioplasty (CP) are surgical procedures used to manage elevated intracranial pressure (ICP) in various clinical scenarios, including ischemic stroke, hemorrhagic stroke, and traumatic brain injury. The physiological changes following DCE, such as cerebral blood flow, perfusion, brain tissue oxygenation, and autoregulation, are essential for understanding the benefits and limitations of these procedures. A comprehensive literature search was conducted to systematically review the recent updates in DCE and CP, focusing on the fundamentals of DCE for ICP reduction, indications for DCE, optimal sizes and timing for DCE and CP, the syndrome of trephined, and the debate on suboccipital CP. The review highlights the need for further research on hemodynamic and metabolic indicators following DCE, particularly in relation to the pressure reactivity index. It provides recommendations for early CP within three months of controlling increased ICP to facilitate neurological recovery. Additionally, the review emphasizes the importance of considering suboccipital CP in patients with persistent headaches, cerebrospinal fluid leakage, or cerebellar sag after suboccipital craniectomy. A better understanding of the physiological effects, indications, complications, and management strategies for DCE and CP to control elevated ICP will help optimize patient outcomes and improve the overall effectiveness of these procedures.

**Keywords:** Decompressive craniectomy; Intracranial pressure; Craniotomy; Craniocerebral trauma

## INTRODUCTION

In recent years, the field of neurocritical care has witnessed significant advancements in the understanding and application of decompressive craniectomy (DCE) and cranioplasty (CP) as critical therapeutic interventions for various neurological conditions. This special issue aims

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**Conflict of Interest**

The authors have no financial conflicts of interest.

to provide a comprehensive update on the physiological effects, indications, complications, and management strategies associated with DCE and CP. We begin by examining the fundamentals of DCE for intracranial pressure (ICP) reduction, focusing on the Monro-Kellie Doctrine and its implications in clinical practice. Furthermore, we discuss the complex physiological changes following DCE, including alterations in cerebral blood flow (CBF), perfusion, brain tissue oxygenation, and autoregulation. The issue also addresses the specific indications for DCE, such as ischemic stroke, hemorrhagic stroke, and traumatic brain injury (TBI), while shedding light on the optimal size of DCE and the timing of CP to achieve the most favorable outcomes. Additionally, we explore the Syndrome of Trephined (SoT) and its association with changes in CBF and neurological symptom improvement following CP. Lastly, we delve into the ongoing debate surrounding the necessity of suboccipital CP, particularly in cases involving persistent headaches, cerebrospinal fluid (CSF) leakage, or cerebellar sag after suboccipital craniectomy (SOC). Through an in-depth analysis of these topics, this issue seeks to enhance our understanding of DCE and CP and their role in the ever-evolving landscape of neurocritical care.

## METHOD

To systematically narrative review the recent updates in DCE and CP, we conducted a comprehensive literature search using the following electronic databases: MEDLINE, Embase, and Cochrane Library. The search terms included “decompressive craniectomy,” “cranioplasty,” “intracranial pressure,” “ischemic stroke,” “hemorrhagic stroke,” “traumatic brain injury,” “optimal size,” “optimal timing,” “syndrome of trephined,” and “suboccipital cranioplasty.” The search was limited to articles published between January 2010 and December 2022, and only articles written in English were considered for inclusion.

Two independent reviewers screened the search results by title and abstract. Articles were considered for full-text review if they were deemed relevant to the study's objectives. Any disagreements between the reviewers were resolved by consensus or consultation with a third reviewer. Full-text articles were reviewed to determine eligibility for inclusion in the systematic review.

We included studies that reported on physiological changes following DCE, indications for DCE, optimal size of DCE, optimal timing of CP, SoT, and the necessity of suboccipital CP. Both randomized controlled trials and observational studies were considered for inclusion. Case reports, case series, and expert opinions were excluded.

A narrative synthesis of the findings from the included studies was conducted, focusing on the physiological effects, indications, complications, and management of DCE and CP. Due to the heterogeneity in study design and reported outcomes, a meta-analysis was not performed. Instead, we present a descriptive summary of the available evidence, including a discussion of the strengths and limitations of the reviewed studies, and provide recommendations for clinical practice and future research.

### Ethical approval/informed consent

We confirm that, for this work ethical guidelines, ethical approvals (institutional review board) and the use of informed consent were not applicable.


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## LITERATURE REVIEW

### Fundamentals of decompressive craniectomy for intracranial pressure reduction

*ICP and the Monro-Kellie doctrine*

A systematic discussion of ICP and its determining factors began in the early 18th century with Scottish anatomist Alexander Monro and his colleague, surgeon George Kellie. The Monro-Kellie doctrine on ICP was later refined by American neurosurgeon Harvey Cushing, providing a detailed explanation of the principles underlying changes and adaptations in ICP. The skull vault is essentially a fixed structure; thus, the volume within the cranial cavity remains constant under normal conditions, with ICP maintained through a balance of its contents. These contents comprise 1) brain tissue, 2) blood, and 3) CSF. Typically, brain tissue occupies a volume of 1,469±102 $cm^3$ in males and 1,289±111 $cm^3$ in females, while CSF amounts to 90–150 mL (with a production rate of 0.2–0.7 mL/min or 500–700 mL/day), and blood occupies 100–130 mL (15% arterial, 40% venous, and 45% in microcirculation) (**FIGURE 1**).[23,77]

However, brain tissue is incompressible; thus, to maintain constant ICP, compensatory adaptations through the influx and efflux of fluid components (blood and CSF) are necessary. In other words, mechanisms for arterial blood inflow, venous blood outflow from the cranial cavity, and adaptations between CSF production and drainage must operate. Consequently, an increase in ICP can arise from any mechanism that augments the volume of these three

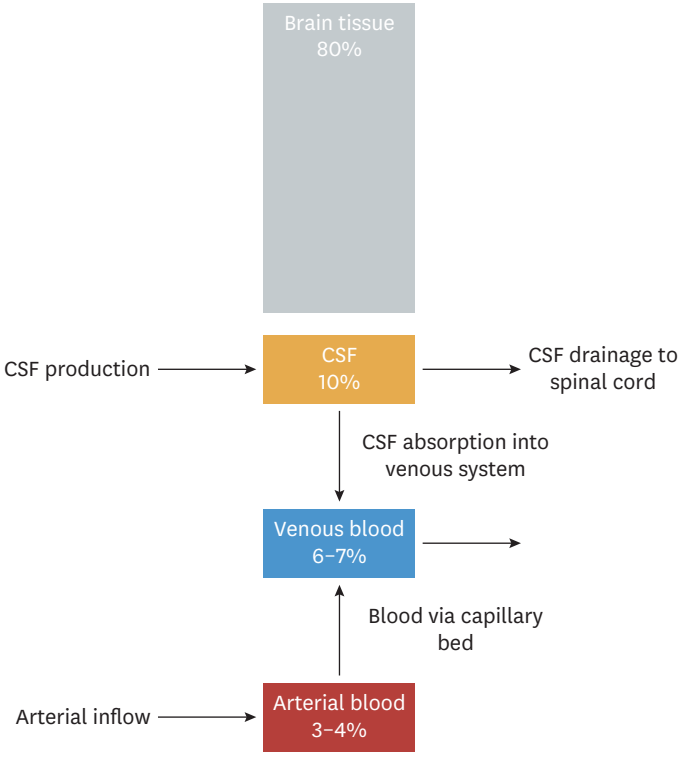


**FIGURE 1.** The Monro-Kellie model illustrating the components of the intracranial compartment. This model demonstrates the delicate balance between brain tissue, CSF, and blood within the rigid skull. 'Brain tissue' encompasses neurons, glia, extracellular fluid, and cerebral microvasculature. 'Venous' and 'arterial blood' represent the intracranial blood volume in macrovasculature and cerebral venous sinuses. 'CSF' includes ventricular and cisternal CSF.
CSF: cerebrospinal fluid.


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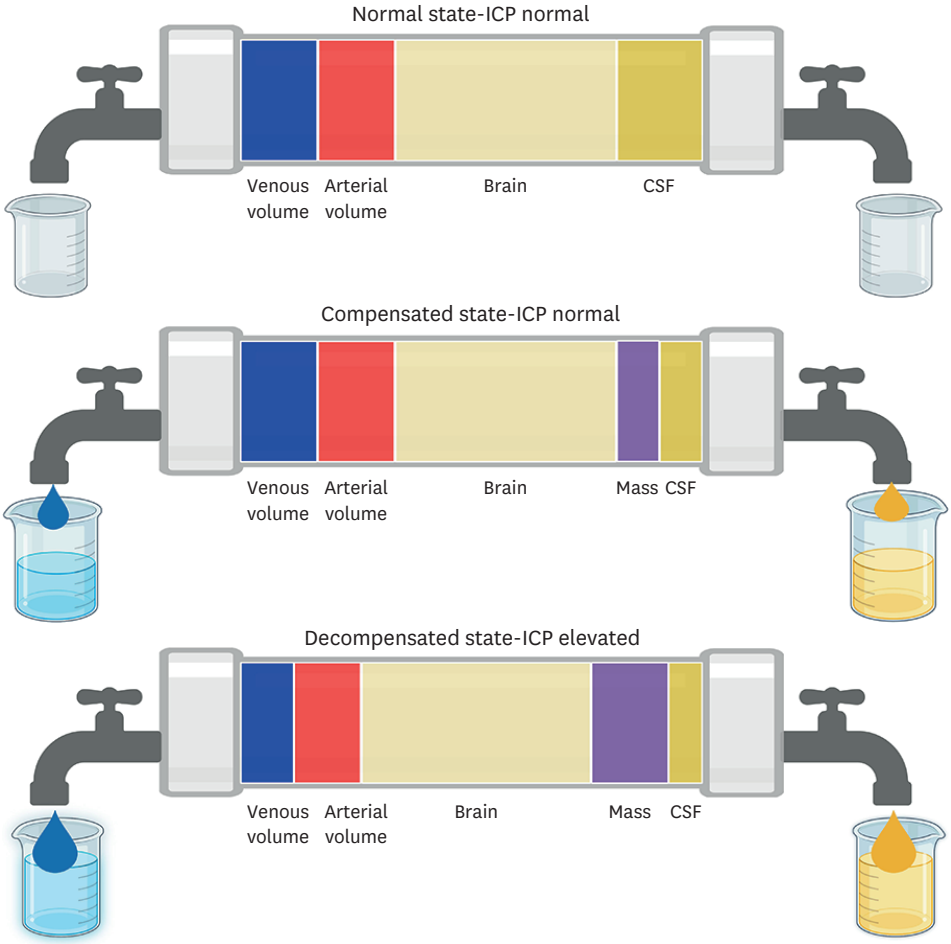


**FIGURE 2.** The Monro-Kellie Doctrine illustrating intracranial compensation mechanisms in response to an expanding mass.[1] This doctrine demonstrates the complex interplay between brain tissue, CSF, and blood within the confined space of the skull. According to the Monro-Kellie Doctrine, when an expanding mass is introduced, compensatory mechanisms involving the reduction of one or both other components are employed to maintain constant intracranial pressure. These compensatory changes can reach a limit, after which further increases in mass may lead to rapid elevation of intracranial pressure and potential brain herniation.
ICP: intracranial pressure, CSF: cerebrospinal fluid.

constituents (brain tissue, blood, and CSF). While the cranial cavity can compensate for an increase in brain content due to edema, intracranial hemorrhage, or brain swelling by decreasing the volume of other components within its confined space, ICP will increase once this compensatory limit is exceeded (**FIGURE 2**).[2,3] The increase in ICP remains constant up to a certain volume, but beyond the compensatory threshold, it increases exponentially (**FIGURE 3**). Hence, it is crucial to actively implement ICP reduction before exceeding this threshold.

DCE can be performed to control pathologically elevated ICP. DCE, which operates on the premise that ICP forms due to the presence of brain tissue, blood, and CSF within the rigid skull, breaks this precondition by expanding the confined cranial space. This method is considered for maintaining ICP in various elevated pressure situations. However, DCE should only be considered when ICP cannot be maintained even with the most aggressive medical therapy. Initial ICP management should include elevating the head up to 30 degrees, administering sedatives and analgesics, maintaining $PCO_2$ at 38–40 mmHg, maintaining normothermia, administering hyperosmolar drugs (mannitol or hypertonic saline),


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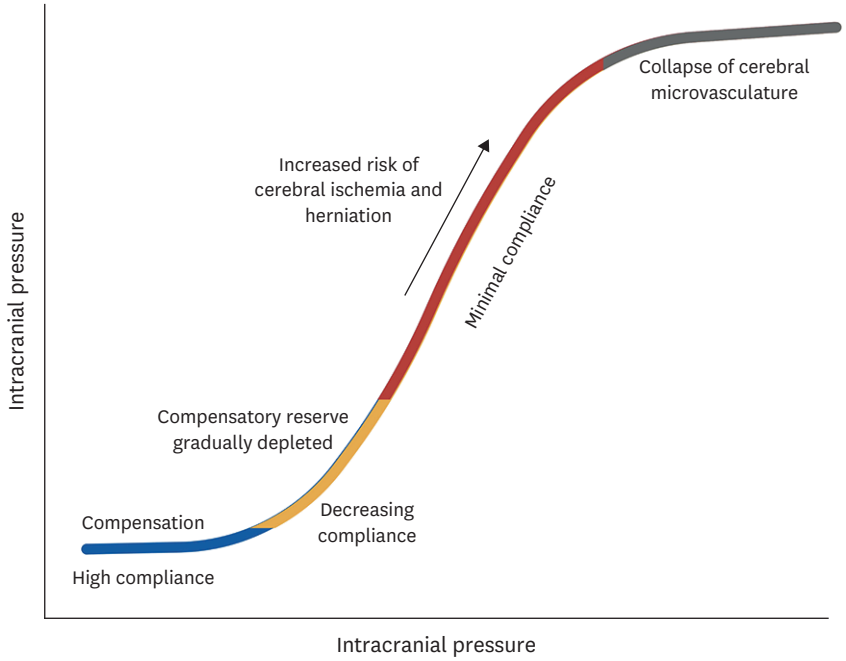


**FIGURE 3.** Pressure-volume curve for ICP.[1,2] This curve demonstrates the relationship between intracranial volume and ICP, with four distinct 'zones': (1) baseline intracranial volume with good compensatory reserve and high compliance (blue); (2) gradual depletion of compensatory reserve as intracranial volume increases (yellow); (3) poor compensatory reserve and increased risk of cerebral ischemia and herniation (red); and (4) critically high ICP causing collapse of cerebral microvasculature and disturbed cerebrovascular reactivity (grey). The curve highlights the importance of monitoring and managing ICP in clinical practice, as changes in intracranial volume can have significant implications for patient outcomes.
ICP: intracranial pressure.

preventing hyperglycemia, and maintaining adequate cerebral perfusion pressure (CPP). Additionally, CSF drainage through ventriculostomy can be considered, and if DCE is difficult to perform, barbiturate coma therapy or hypothermia therapy can be initiated preoperatively.

Pharmacological treatment can be used for ICP values of 20–25 mmHg when CPP or brain tissue oxygen tension ($PbtO_2$) is deemed adequate, without resorting to DCE. However, if persistent ICP elevation remains uncontrolled despite the aforementioned methods or if a decrease in CPP or $PbtO_2$ is observed or anticipated, DCE should be considered.[24]

*Decompressive effect of decompressive craniectomy (DCE) and duroplasty*

Despite maximum pharmacological treatment, if ICP is not adequately controlled, decreased CPP due to elevated ICP can lead to ischemic cell damage and death, resulting in secondary injuries such as ischemic brain injury and cerebral edema.[78] In patients with uncontrolled increased ICP, DCE can be considered to aid in ICP control. Evidence supporting DCE shows a direct relationship between the total time of ICP elevation above 20 mmHg and poor prognosis.[61] Prolonged uncontrolled ICP is associated with increased mortality and poorer neurological outcomes.[61] Therapeutic approaches to reduce ICP include decreasing the volume of intracranial contents (blood, brain tissue, or CSF), reducing cerebral metabolic demand (hypothermia treatment, barbiturate therapy, etc.), and increasing intracranial voluminal capacity through DCE. The DCE not only shifts the pressure-volume curve to the right, reducing the amplitude of ICP waves, but also improves intracranial compliance (**FIGURE 4**).[78]


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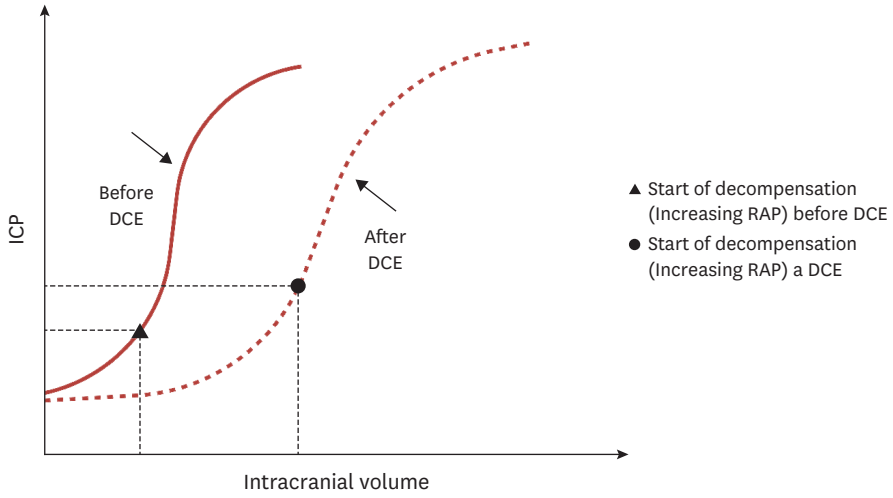


**FIGURE 4.** Pressure-volume curve for ICP before and after decompressive craniectomy.[6,7] The curve illustrates the relationship between intracranial volume and ICP, emphasizing the impact of DCE on the pressure-volume compensatory reserve (RAP). The red line represents the pressure-volume curve before decompressive craniectomy, while the red dot-line indicates the pressure-volume curve after decompressive craniectomy. Following decompressive craniectomy, there is an increase in the pressure-volume compensatory reserve, as demonstrated by the shift in the curve. This figure highlights the effectiveness of decompressive craniectomy in alleviating elevated ICP and improving intracranial compliance.
DCE: decompressive craniectomy, ICP: intracranial pressure, RAP: pressure-volume compensatory reserve.

Following DCE, the reduction in ICP allows for an increase in CBF, CPP, and cerebral microcirculation, enabling the reestablishment of the balance between cerebral inflow and outflow. Brain tissue oxygenation ($PbtO_2$) also improves. Thus, DCE reduces ICP, and decreased ICP can improve both survival rates and neurological prognosis.

The reduction of ICP through DCE has been demonstrated in several studies. The mean ICP before skin incision was 41±16.2 mmHg, which decreased to an average of 18 mmHg after DCE. In cases where duroplasty was also performed, ICP decreased further to an average of 10.6 mmHg (**FIGURE 5**).[5,43] Additionally, in several studies, DCE effectively reduced ICP and was beneficial for maintaining CPP. Significant ICP reduction has also been reported in patients who underwent DCE compared to those who received medical treatment alone for head trauma.[46,60,71]

However, in the DECRA trial conducted in head trauma patients, DCE reduced ICP and decreased the duration of intensive care unit stays but resulted in a higher proportion of patients with poor functional outcomes. These results for TBI suggest the presence of additional pathophysiological changes (atmospheric pressure, CSF dynamic changes, venous drainage changes, transcapillary leakage, etc.) that occur in decompressive surgery, which may compromise the benefits of ICP reduction and cerebral perfusion improvement.[60] In contrast, the DESTINY II trial showed that DCE increased survival rates and had favorable functional outcomes in patients with uncontrolled ICP elevation accompanied by cerebral infarction.[32] The RESCUEicp trial also reported favorable survival rates and functional outcomes in patients with TBI who underwent DCE.[28]

*Physiological changes following DCE improvement of cerebral blood flow, perfusion, and brain tissue oxygenation, autoregulation following DCE*

Following DCE, the intracranial space expands and ICP decreases, leading to changes in CBF and CSF circulation. Although the reduction in ICP generally improves CBF and CSF


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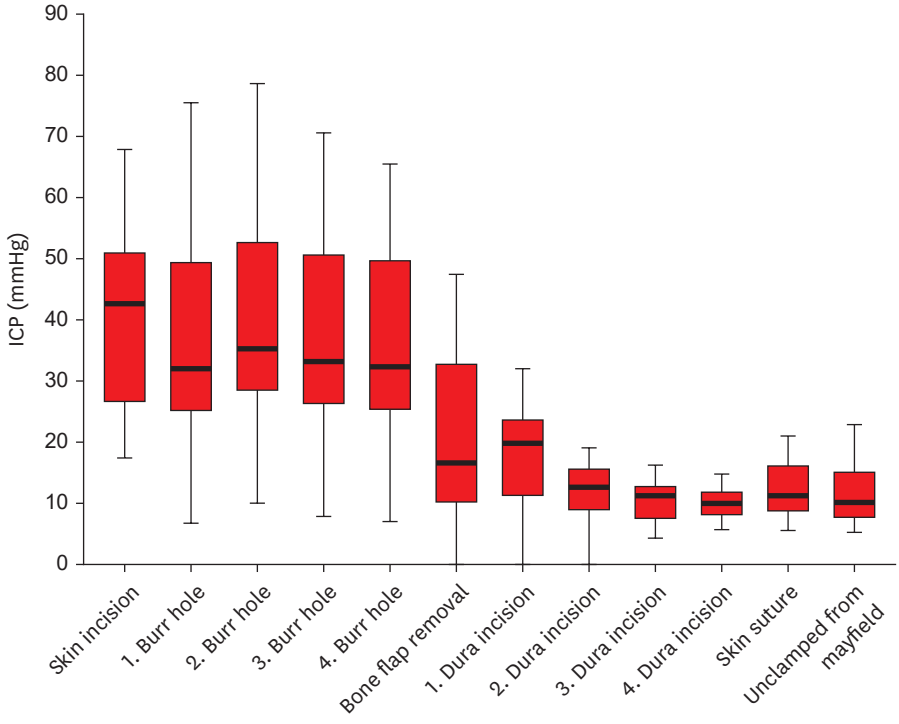


**FIGURE 5.** ICP values at the each surgical steps[6,7] The box plot illustrates the median ICP values (solid line within each box) and the interquartile range (p25 to p75) represented by the boxes. This figure highlights the variations in ICP throughout the surgical process, emphasizing the importance of each surgical procedures to decrease ICP during neurosurgical procedures to optimize patient outcomes.
ICP: intracranial pressure.

circulation, thereby ameliorating clinical outcomes, it does not exclusively produce favorable effects. In patients with TBI, CBF increased in the decompressed brain lesions within 24 hours after the craniectomy. This finding was confirmed by evaluating the changes in regional cerebral blood flow (rCBF) using single-photon emission computed tomography performed immediately before surgery, immediately after surgery (within 24 hours), one week later, and one month later. Immediately after surgery, low perfusion areas were clearly surrounded by hyperperfusion areas in the decompressed hemisphere. One week later, both the severity and size of the hyperperfusion areas increased. After one month, hyperperfusion appeared to subside. Hyperperfusion areas coincided with areas of cerebral edema on brain CT scans. The authors attributed the increased rCBF and cerebral edema to cerebrovascular dysregulation and increased CPP following DCE.[38]

Perfusion CT analysis after DCE revealed increased CBF and cerebral blood volume (CBV) not only in the lesion but also in the contralateral hemisphere.[66] In another study, cerebral microvascular blood flow doubled after DCE, as measured by contrast-enhanced ultrasound performed before and after surgery. This increase was primarily due to an increase in microvascular blood volume. CBF and CBV increased on average by five times until the third day after surgery. Such increased blood flow can exacerbate cerebral edema, and when combined with increased postoperative CPP, impaired cerebral vascular pressure reactivity, and cerebral inflammation, there is a possibility of cerebral edema. By controlling CBF, CPP, ICP, and arterial blood pressure (ABP) within appropriate ranges after surgery, the potential exacerbation of cerebral edema can be prevented.[7]


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Along with increased CBF following DCE, indicators of brain metabolism, such as brain tissue oxygenation ($PbtO_2$) and microdialysis, also improved. In patients with subarachnoid hemorrhage (SAH), $PbtO_2$ significantly increased from hypoxic levels (6 mmHg) to normal oxygen levels (23 mmHg) following DCE and a decrease in ICP.[38] In another study, $PbtO_2$ increased from an average of 21.2±13.8 to 45.5±25.4 mmHg in patients with TBI and SAH following DCE and increased CBF.[38]

However, there are conflicting results regarding the indicators representing cerebral autoregulation. The pressure reactivity index (PRx), which represents the dynamic response of ICP to changes in ABP, is an indicator of cerebrovascular reactivity. Normally, responsive cerebral arteries contract in response to a systemic increase in arterial pressure, resulting in a transient decrease in CBV and a subsequent decrease in ICP. Thus, in cases with normal cerebral autoregulation, changes in blood pressure and ICP exhibit a negative correlation. However, in patients with impaired cerebral autoregulation, changes in arterial pressure and ICP simultaneously increase, showing a positive correlation. Following DCE, ICP decreases, but arterial pressure and ICP may still exhibit a positive correlation. This suggests that the autoregulation function (PRx) might actually decrease after DCE.[71] The mechanism for this is not yet clear, but it is hypothesized to be related to the expansion of brain tissue after DCE, leading to a decrease in brain elasticity, as well as changes in the autoregulatory function of the brain tissue and cerebral vessels exposed to atmospheric pressure. Additionally, some studies have shown that although ICP reduction is achieved after DCE, the cerebral metabolic rate of O2 remains unchanged.[38] These findings are particularly pronounced in patients who exhibit poor prognosis following DCE.[73] Thus, while the ICP reduction effect has been demonstrated after DCE, further research on additional hemodynamic and metabolic indicators is warranted.

## Indications for DCE

*Ischemic stroke*[56,57]

In patients with cerebral infarction, DCE is performed to prevent secondary brain damage caused by cerebral edema and increased ICP due to ischemic stroke. Hemicraniectomy is performed for cerebral infarctions, while SOC is conducted for cerebellar infarctions.

DCE is performed in patients with cerebral infarction to relief malignant cerebral edema, increased ICP, and subsequent brain herniation resulting from extensive ischemic areas. The effectiveness of DCE in reducing ICP caused by cerebral edema is well-established. Several studies have reported that DCE reduces mortality rates by approximately 50% in patients with cerebral edema due to unilateral middle cerebral artery (MCA) infarction compared to pharmacological treatment, making it an essential life-saving treatment.[17,18,33,34,49,72] However, patients with MCA infarction have a high mortality rate and a high likelihood of living with severe disabilities due to neurological sequelae. Therefore, functional recovery should also be considered beyond merely reducing mortality rates. The AHA/ASA guidelines[56] recommend considering these characteristics when deciding on DCE and provide varying levels of recommendations based on age and symptom onset time based on the DECIMAL,[72] DESTINY I,[33] II,[32] HAMLET,[18,26] Zhao et al.,[80] and HeADDFIRST[17] randomized controlled trials (RCTs). The DECIMAL,[72] DESTINY I,[33] HAMLET[18] RCTs, and three pooled meta-analyses demonstrated reduced mortality and severe disability (mRS 5–6) in patients aged 60 and below, while the DESTINY II[32] and Zhao et al.[80] RCTs showed reduced mortality and severe disability (mRS 5–6) in patients aged 80. However, the functional outcomes were significantly worse compared to those under 60 years of age. Based on these findings, the


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**TABLE 1.** Overview of the randomized controlled trials[50] about decompressive craniectomy for cerebral infarction[28]

| Study name | Age (years) | Inclusion from symptom onset (hours) | Imaging criteria | Clinical criteria | Primary outcome parameter | Main finding |
|---|---|---|---|---|---|---|
| DECIMAL | 18–55 | <24 | >50% ischemic MCA territory; MRI-DWI infarct volume >145 cc | NIHSS>15; NIHSS 1a≥1 | mRS 0–3 at 6 months | 52.5% absolute mortality reduction with DC compared to BMT($p$<0.0001); no significant difference between DC and BMT regarding mRS 0–3 |
| DESTINY I | 18–60 | >12 to <36 | ≥2/3 MCA territory with basal ganglia; with/without ACA/PCA territory | NIHSS>18(non-dominant) or >20 (dominant); NIHSS 1a≥1 | Sequential design; mortality after 30 days; mRS 0–3 vs. 4–6 at 6 months | Mortality reduction from 88% to 47% with DC after 30 days ($p$=0.02) |
| HAMLET | 18–60 | <96 | ≥2/3 MCA territory; formation of space occupying edema | NIHSS ≥16(right) or ≥21 (left); NIHSS 1a ≥1; GCS <13 (right-sided) or GCS (eye and motor score) <9 (left-sided) | mRS 0–3 vs. 4–6 at 12 months | DC with no effect on primary outcome measure but significant reduction of case fatality (ARR 38%) |
| Zhao et al. | 18–80 | <48 | ≥2/3 MCA territory | GCS (eye and motor score) ≤9 | mRS 0–4 vs. 5–6 at 6 months | Reduction of mortality (DC 12.5% vs. BMT 60.9%, $p$=0.001) and mRS 5–6(DC 33.3% vs. BMT 82.6 %, $p$=0.001) |
| HeADDFIRST | 18–75 | <96 | ≥50% ischemic MCA territory (<5 hours) or complete MCA infarction (<48 hours) | NIHSS ≥18; NIHSS 1a <2 | Survival 21 days | Non-significant reduction of mortality at 21 days (DC 21% vs. BMT 40%, $p$=0.39) |
| DESTINY II | >60 | <48 | ≥2/3 MCA territory with basal ganglia | NIHSS >14 (non-dominant) or >19 (dominant), reduced level of consciousness on NIHSS | mRS 0–4 at 6 months | Significant reduction of severe disability (mRS scroes 5–6; DC 38% vs. BMT 18%, $p$=0.04) |

MCA: middle cerebral artery, MRI: magnetic resonance imaging, DWI: diffusion weighted image, NIHSS: National Institute of Health Stroke Scale, mRS: modified Rankin Scale, DC: decompressive craniectomy, BMT: best medical treatment, ARR: absolute risk reduction.

guidelines recommend Class IIa, Level A for patients aged 60 and below, and Class IIb, Level B for those over 60 years of age when neurological deterioration occurs due to cerebral edema from unilateral MCA infarction, DCE should be conducted within 48 hours of symptom onset (**TABLE 1**).[56]

Regarding the timing of decompressive surgery, the most optimal time is unknown, but it is recommended to consider surgery when the patient exhibits a decreased level of consciousness (Class IIa, Level A).[56] Generally, cerebral edema peaks between 2–5 days after the onset of cerebral infarction, and neurological deterioration due to this edema occurs within 48 hours. Although the timing of decompressive surgery varies among studies, research has been conducted within 4 days, including at 36, 48, and 96 hours. The HAMLET study investigated DCE timing within 96 hours of symptom onset and found that patients who underwent surgery within 48 hours had reduced mortality rates (19% vs. 78%) and severe disability rates (mRS 5–6; 48% vs 78%).[26] Based on these findings, early DCE within 48 hours of symptom onset after cerebral infarction is recommended.[18,26]

The extent of cerebral infarction is also an important factor in determining to perform DCE. According to Sundseth et al.'s study[69] on early mortality prediction in patients who underwent DCE for cerebral edema caused by MCA infarction, early mortality rates were significantly higher when the infarction progressed to the anterior cerebral artery (ACA) territory (42.9% vs 19.4%). Therefore, in patients with cerebral infarction involving the ACA territory, the decision to perform DCE should be made early, and this factor should be considered when determining the timing of surgery.

Cerebellar infarction-induced cerebral edema can cause rapid neurological deterioration due to brainstem compression and the development of acute obstructive hydrocephalus,


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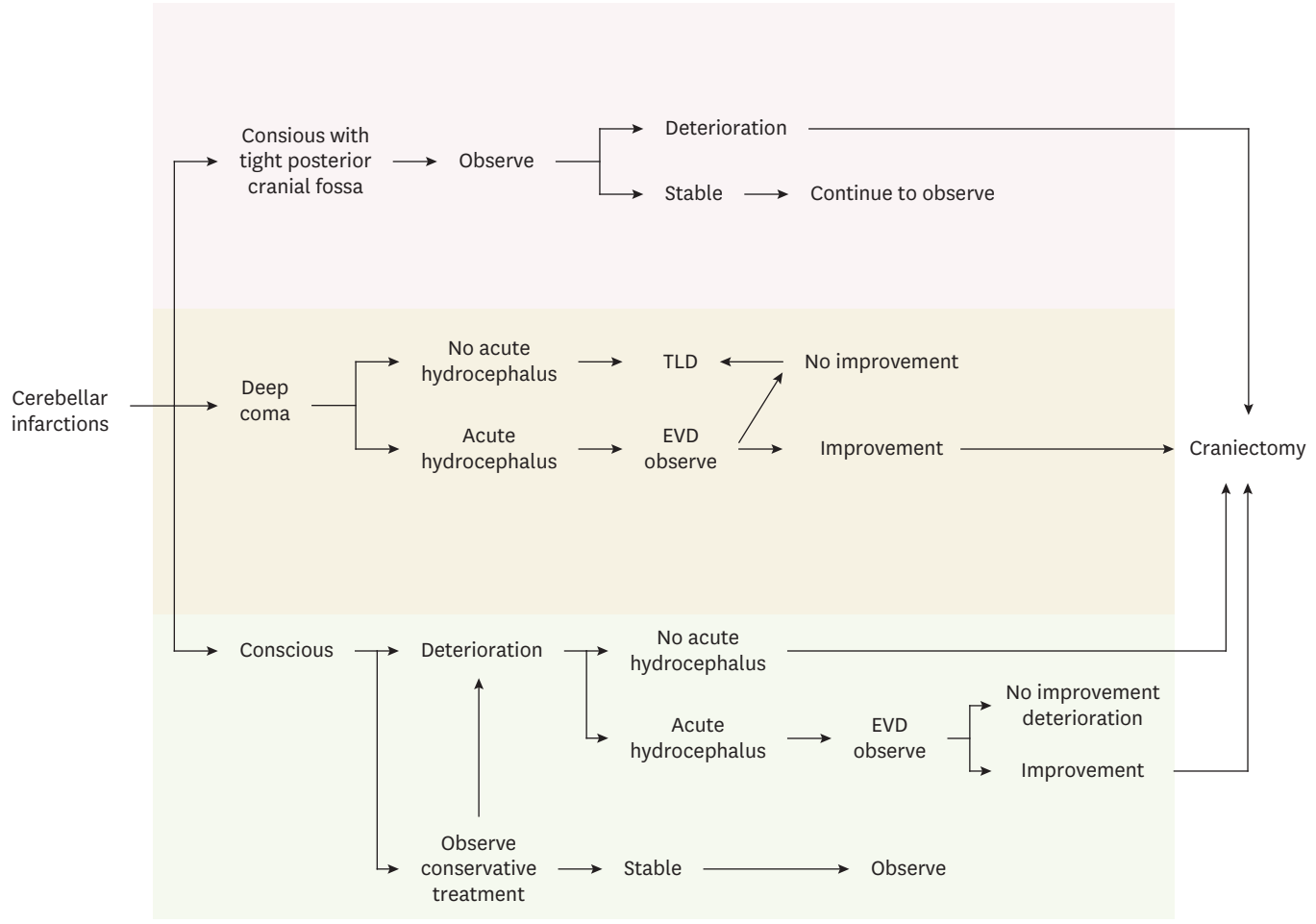


**FIGURE 6.** Treatment algorithm for cerebellar infarction.
EVD: extraventricular drainage, TLD: treat-limiting decision.

stemming from the anatomical structure of the inferior tentorium. Extraventricular drainage (EVD) is an effective treatment for obstructive hydrocephalus and can bring about significant neurological improvements in patients. However, when brainstem compression occurs due to cerebellar edema, EVD alone is insufficient, and SOC and decompression through the removal of dead brain tissue, if necessary, are required. Therefore, when neurological deterioration occurs, it is crucial to determine the presence of hydrocephalus, brainstem compression, and whether SOC is necessary in addition to EVD. In this regard, the AHA/ASA guidelines recommend performing EVD if obstructive hydrocephalus is present, and initiating SOC from the outset if there is no response to EVD or if brainstem compression occurs due to mass effect (Class I) (**FIGURE 6**).[56,58]

*Hemorrhagic stroke*

In spontaneous intracerebral hemorrhage (sICH), recommendations are similarly divided into cerebral hemorrhage and cerebellar hemorrhage, as with cerebral infarction. Despite of the high incidence of brainstem compression and acute obstructive hydrocephalus caused by the anatomical characteristics of the suboccipital tentorium, randomized studies are limited. However, hematoma evacuation is recommended for patients with neurological deterioration or those with hemorrhages larger than 15mL to reduce mortality rates compared to medical


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treatment alone. Patients who underwent SOC, C1 posterior arch removal for decompression, and hematoma removal in the cerebellar hemorrhage showed better neurological outcomes compared to those who only underwent hematoma removal surgery, but the results were not statistically significant.[21] Therefore, further research is needed to determine whether performing skull decompression simultaneously with hematoma removal is more beneficial.

For large ICH located in the supratentorial region, DCE is considered due to neurological deterioration, midline shift, and increased ICP. Many studies have shown that DCE reduces ICP and mortality rates, but there is insufficient evidence for functional improvement.[52,79] Most studies involve patients in a coma (GCS<8), those with large hematomas (>30 mL), and those with persistently elevated ICP despite medical treatment. Surgical methods can be divided into cases where only DCE was performed, only hematoma removal was performed, or both were performed simultaneously.

According to a systematic review, mortality rates in patients with sICH were lower in the group that underwent DCE compared to the group that received only medical treatment (26% vs. 51%), but there was no difference in functional outcomes.[52] In patients with deep ICH there was no significant difference in mortality or functional outcomes between those who underwent simultaneous DCE and hematoma removal and those who only underwent DCE.[58] In some RCT studies, although there was no significant difference in mortality rates between patients who underwent only hematoma removal and those who underwent simultaneous DCE, hematoma removal, and duroplasty, the latter group showed favorable functional outcomes (mRS 0–3 at 6 months; 70% vs. 20%), indicating good functional recovery.

In patients with SAH caused by a ruptured aneurysm, a systematic review and meta-analysis of DCE showed that younger age, good grade SAH, and primary DCE were associated with better outcomes, suggesting that DCE can be considered in selected patient populations.[13]

Based on these studies, DCE in patients with hemorrhagic stroke has the favorable effect of reducing mortality rates, but the effect on neurological functional improvement is unclear. Therefore, especially in patients with large supratentorial hematomas with persistent ICP elevation and midline shift despite medical treatment, DCE should be considered for life-saving purposes to reduce mortality rates. The effect on neurological functional improvement is uncertain, so surgical decisions should be made considering these factors (Class 2b, Level C-LD).

*TBI*[9,10,25]

In patients with TBI, there is a lack of Level I evidence for DCE. There are two RCTs comparing the effects of medical treatment and DCE in TBI patients: the DECRA[11] trial and the RESCUEicp[28] trial. In the DECRA[11] trial, DCE was performed within 72 hours (early) in cases where conservative treatment was ineffective (based on an ICP of 20 mmHg), while in the RESCUEicp[28] trial, DCE (bilateral or unilateral DCE) was performed regardless of time, when ICP >25 mmHg persisted for more than 1 hour after conservative treatment. Both studies demonstrated that DCE effectively reduced ICP and decreased mortality rates; however, functional recovery was not observed.

Based on these results, bifrontal DCE was not recommended in 2017 for the purpose of improving functional outcomes (GOSE at 6 months). However, considering the findings of the DECRA trial analyzing 12-month outcomes and the results published in the RESCUEicp trial, the 2020 recommendations were updated to do DCE in patients with elevated ICP


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unresponsive to medical treatment for improving mortality and favorable outcomes (Level IIA).[25] Although the direct relationship between the effects of DCE and favorable outcomes remains unclear, it is now recommended to perform DCE (Level IIA) as it lowers ICP and reduces the length of stay in the intensive care unit.[10,25]

In 2022, the results of a secondary analysis of the RESCUEicp trial at 24 months were published, showing that, at 24 months, the patients who underwent DCE had reduced mortality rates compared to those who received only medical treatment.[35] However, there were higher rates of vegetative state, severe, and moderate disability, but a significant improvement of grade 1 or higher was observed between 6 and 24 months. Furthermore, the RESCUE-ASDH trial is currently underway, investigating the role of DCE in patients with acute traumatic subdural hematoma. Therefore, it is necessary to closely monitor future research findings on the role of DCE in patients with TBI (**TABLE 2**).

## Optimal size of DCE

The importance of DCE for TBI patients has been demonstrated in the DECRA[11] and RESCUEicp[28] studies. In unilateral DCE, the size of the bone flap removal is a critical factor in determining patient prognosis.[36] Recent guidelines recommend larger fronto-temporo-parietal decompressive craniectomies (diameter 12×15 cm or >15 cm) over smaller ones to achieve better mortality rates and functional outcomes.[10] A study involving TBI patients showed that the group that underwent standard DCE (12x15 cm) had better functional outcomes than the group that underwent limited DCE (6×8 cm) (39.8% vs. 28.6%).[31] Furthermore, the mortality rate was also lower in the standard DCE group compared to the limited DCE group (26.2% vs. 35.1%).

In another study analyzing the relationship between the size of the DCE and patient prognosis in 20 TBI patients, the relationship between craniectomy size and mortality was significant, but the significance with functional outcomes could not be established.[63] Notably, all patients with craniectomy diameters less than 10 cm died. One study analyzed a group of 74 patients who underwent very large decompressive craniectomies (≥12 cm).[70] When analyzed in three categories, 12–15 cm, 15–20 cm, 20–24 cm, no differences in outcomes were observed. This study found that the risk of complications such as bleeding or cerebral edema did not increase in the group that underwent 12 cm DCE compared to the

**TABLE 2.** Summary of recommendation of decompressive craniectomy in ischemic stroke, hemorrhagic stroke, and traumatic brain injury

| Disease | Site | Recommendation |
|---|---|---|
| Ischemic stroke | Supra-tentorial | ≤60, onset within 48 hours, unilateral MCA infarction with refractory brain swelling: DCE with dural expansion is reasonable (Class IIa, Level A). |
| | | >60, onset within 48 hours, unilateral MCA infarction with refractory brain swelling: DCE may be considered (Class IIb, Level B). |
| | Infra-tentorial | Ventriculostomy is recommended in obstructive hydrocephalus after cerebellar infarction. Concomitant or subsequent decompressive craniectomy may be necessary (Class I, Level C). |
| | | Cerebellar infarction causing neurological deterioration from brainstem compression despite maximal medical therapy: Decompressive SOC should be performed (Class I, Level B). |
| Hemorrhagic stroke | Supra-tentorial | In a coma (GCS<8), large hematoma (>30 mL) with significant midline shift, refractory IICP: DCE may be considered to reduce mortality but effectiveness of DCE to improve functional outcomes is uncertain (Class 2b Level C). |
| | Infra-tentorial | ≥15 mL, ventricular obstruction: immediate surgical removal of ICH is recommended to reduce mortality (Class I, Level B). |
| | SAH | Clinical evidence on the use of DCE in aSAH is limited but due to expected outcome benefit, DCE may be considered in case of young and good grade with IICP. |
| TBI (Level IIA) | | Secondary DCE performed for late refractory ICP elevation is recommended to improve mortality and favorable outcomes. |
| | | Secondary DCE performed for early refractory ICP elevation is not recommended to improve mortality and favorable outcomes. |
| | | Secondary DCE performed as a treatment for either early or late refractory ICP elevation is suggested to reduce ICP and duration of ICU |

MCA: middle cerebral artery, DCE: decompressive craniectomy, SOC: suboccipital craniectomy, GCS: glasgow coma scale, IICP: increased intracranial pressure, SAH: subarachnoid hemorrhage, TBI: traumatic brain injury, ICU: intensive care unit.


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group that underwent larger craniectomies. Therefore, considering these results, it appears that the effect of DCE can be seen when the size of the craniectomy reaches around 12 cm, but an increase in size does not necessarily improve the prognosis.

### Optimal timing of CP

There is still controversy regarding the optimal timing of CP following DCE. The timing varies depending on the clinical indications for craniectomy. For cases with acute intracranial hypertension, CP should be performed after adequate control of ICP. However, in other cases (chronic intracranial hypertension due to brain tumors or skull lesions), immediate CP can be performed. Ultimately, the timing of CP should consider the patient's condition and be performed after adequate control of ICP and minimizing the risk of infections. This article aims to discuss the timing of CP following ICP control after DCE for acute intracranial hypertension.

There is no clear criterion for dividing the timing of early CP and late CP, but many studies define it based on a 3-month period.[1,44] Traditionally, CP has been performed 3 months after DCE to allow for sufficient neurological and systemic recovery. However, recent research suggest that early CP (within 3 months) may facilitate CSF dynamics recovery and be advantageous for neurological recovery.[12,55,74,75] Moreover, from a technical perspective, early CP at 5–8 weeks post-craniectomy has the advantage of easy tissue layer dissection due to less severe adhesion.[4] On the other hand, late CP is associated with significantly longer surgery time due to tissue adhesion.[54]

Various complications may arise after CP, such as infection, hydrocephalus, hygroma, seizures, intracranial hemorrhage, bone resorption, flap depression, and wound dehiscence. These complications should also be considered when determining the timing of CP. The optimal timing for CP should minimize surgical risks, prevent neurological deterioration due to the craniectomy state, and maximize neurological improvement.

Morton et al. confirmed favorable outcomes in terms of infection, seizure, hematoma, and bone resorption complications in 754 patients who underwent early CP between 15 and 90 days post-craniectomy. However, ultra-early CP (within 14 days) was associated with the highest incidence of hydrocephalus and infection, while late CP (after 90 days) had a higher incidence of seizures.[44] There were research that early CP in patients with TBI significantly increased the incidence of hydrocephalus.[41] However, conflicting research findings exist. A meta-analysis conducted solely on TBI patients reported a significantly lower incidence of hydrocephalus in those who underwent early CP.[45] By performing early CP, excluding the ultra-early phase when CSF dynamics are unstable, normalizing CSF dynamics early on may reduce the risk of hydrocephalus.[45,48] Long-term exposure to the absence of the skull can lead to distribution and absorption disorders of the CSF, increasing the risk of hydrocephalus due to dysfunction of the arachnoid granulation.[45]

Considering the aforementioned research findings, it seems more plausible that early CP does not aggravate hydrocephalus but rather triggers clinical symptoms in patients with underlying hydrocephalus. Therefore, there is no need to delay the timing of CP due to concerns of hydrocephalus development.

Moreover, no statistically significant differences were observed in complications associated with CP, such as intracranial hemorrhage, extra-axial fluid collection, seizures, and bone


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graft resorption, depending on the timing of the procedure. Aseptic osteonecrosis has shown conflicting results in relation to the timing of CP across various studies.[8,16,44] As a result, no significant correlation has been established between the timing of CP and the onset of aseptic osteonecrosis. Numerous studies have already demonstrated that early CP has a positive impact on neurological recovery.[6,27,30,40,47] The most recent meta-analysis, which included eight studies involving 551 patients, reported that CP improved neurological function and that early CP further amplified this effect.[40] The increase in CBF after CP could also be a contributing factor in promoting cognitive recovery.[59,64,76] A study using transcranial Doppler to measure CBF in the MCA and ICA found that the group that underwent early CP had a more significant increase in CBF at the craniectomy site and improved contralateral CBF compared to the group that underwent delayed CP.[67]

Although there is still controversy on the appropriate timing of CP, it can be inferred that performing early CP has beneficial effect in the overall neurological recovery. Specifically, early CP may reduce the risk of hydrocephalus (excluding the ultra-early period before 14 days), facilitate tissue dissection during surgery, and promote neurological function improvement due to the normalization of CSF dynamics and the enhancement of CBF. Therefore, the authors recommend performing early CP within three months after controlling increased ICP.

### SoT/sinking skin-flap syndrome after craniectomy

The SoT, also known as Sinking Skin-Flap Syndrome, is a complication characterized by unexplained neurological deterioration after craniectomy.[2] In 1939, Grant and Norcross first described it as a collection of symptoms, including severe headache, dizziness, pain/discomfort, anxiety, and mental depression after the craniectomy.[20] This syndrome encompasses a wide range of neurological symptoms, including delayed neurological improvement, decreased motor function, cognitive decline, impaired vigilance, and headache.[2] Typically, neurological symptoms caused by SoT can be expected to recover within a short period of 1 to 2 weeks after CP.[2,68] However, there is no consistency in the size of the craniectomy and the duration until CP on the manifestation of SoT symptoms and the degree of improvement after CP. A systemic review of 48 articles revealed that SoT symptoms could appear up to 5 months after craniectomy,[3] and the time to symptom onset varied. Regardless of elapsed time, it is essential for neurocritical care professionals to suspect SoT if the patient has unexplained neurological deterioration. The pathogenesis of SoT is not yet fully understood, but several theories exist (**FIGURE 7**)[3]:

1) Atmospheric pressure: It is argued that atmospheric pressure directly affects the brain at the skull defect site, causing CSF circulation disorders and pressure on cerebral blood vessels, leading to decreased CBF.[2,53] It is assumed that the larger the skull defect, the greater the reduction in blood flow.[14]
2) CBF: The decrease in CBF is due to atmospheric pressure and venous blood flow disturbance, occurring not only at the skull defect site but also on the opposite side.[67,68]
3) CSF flow: Due to skull defects, abnormal CSF circulation can cause brain edema progression and hydrocephalus due to dysfunction of arachnoid granulations.[45,68]
4) Cerebral metabolism: The deterioration of neurological symptoms is explained by cortical dysfunction due to metabolic abnormalities in glucose, as evidenced by the decreased ratio of phosphocreatinine to inorganic phosphate, a marker of brain energy depletion in the damaged hemisphere.[62]


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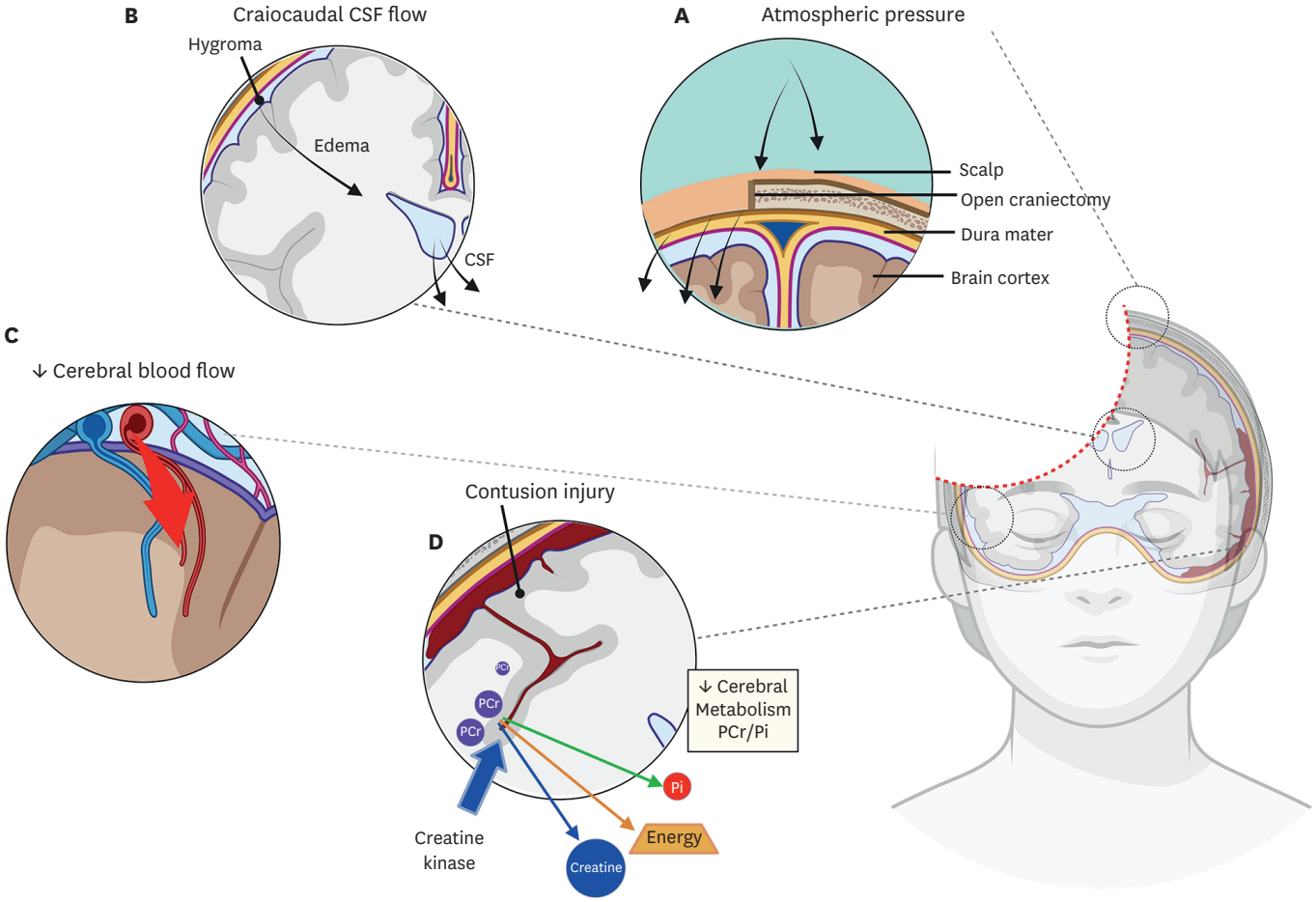


**FIGURE 7.** Illustration demonstrating the four theoretical mechanisms contributing to the syndrome of the trephined. (A) Atmospheric pressure: the absence of the skull bone exposes the brain to external atmospheric pressure, potentially causing deformation and functional disturbances. (B) Craniocaudal CSF flow: alterations in the CSF flow dynamics due to the skull defect may result in abnormal pressure gradients and impaired CSF circulation. (C) Decreased cerebral blood flow: the skull defect may lead to reduced cerebral perfusion, negatively impacting neuronal function. (D) Decreased cerebral metabolism: compromised energy metabolism and oxygen utilization in the affected brain tissue may contribute to the development of neurological symptoms. This figure highlights the complex interplay of factors involved in the pathophysiology of the syndrome of the trephined.
CSF: cerebrospinal fluid, PCr/Pi: phosphocreatine to inorganic phosphate ratio.

Dujovny et al.[15] demonstrated that the systolic CSF flow doubled after CP compared to the craniectomy state, arguing that this change was due to the alleviation of CSF flow obstruction. Additionally, the association between aggravated neurological symptoms in SoT and CBF was proven using cerebral perfusion CT and MRI imaging, and improvements could be observed after CP. The increase in CBF after CP can also promote neurological and cognitive recovery.[59,64,76] A systemic review of 21 articles confirmed that CP improved CBF in a total of 205 cases.[64] A prospective study on patients who underwent craniectomy due to TBI also confirmed that not only improve CBF after CP, but the difference in CBF between the hemispheres also decreased, as demonstrated by CT perfusion performed before and after CP.[76] The improvement of neurological symptoms after CP is also attributed to the normalization of neural tracts. Diffusion tensor imaging was used to compare tractography before and after CP, showing that tracts recovered along with symptom improvement.[29]

### Debate on suboccipital CP: is it necessary?

SOC is a surgical procedure aimed at decompression of lesions in the posterior fossa. During the surgery, most cases involve exposure of the venous sinus or mastoid air cells.


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Furthermore, the procedure often results in a much smaller bone flap than the extent of the craniectomy. Traditionally, SOC has been a permanent skull removal procedure.[51,65] CP for SOC has only been performed more recently, with limited cases until the 1990s, due to concerns of the posterior fossa narrowing again. Leaving the craniectomy open was believed to be safer, easier to perform, more advantageous in controlling brain edema, and associated with lower rates of hydrocephalus. However, recent reports have shown that restoring the bone flap is associated with fewer complications than leaving it removed.[19,22,39]

Failure to reconstruct suboccipital defects can lead to complications such as sinking at the defect site, persistent headaches due to dural adhesion of muscle fibers, pseudomeningocele formation, CSF leakage, arachnoid adhesion, continuous tonsillar compression, and cerebellar sag.[42] Suboccipital CP has been reported to prevent the above complications.

A 2018 survey conducted by the American Association of Neurologic Surgeons[37] revealed significant differences in performing suboccipital CP after posterior fossa decompression, depending on patient characteristics (underlying disease, age), and geographical location. Pediatric cases showed a higher preference for CP, and the proportion of CP performed was higher in brain tumor patients compared to cerebrovascular patients.

Although there has traditionally been a tendency not to perform suboccipital CP following SOC, in patients with persistent headaches, CSF leakage, or cerebellar sag after SOC, it is necessary to positively consider suboccipital CP.

## CONCLUSION

In conclusion, DCE has been regarded as an essential surgical intervention for managing elevated ICP in various clinical scenarios such as ischemic stroke, hemorrhagic stroke, and TBI. However, our understanding of the physiological effects and optimal application of DCE remains an area of ongoing research. While DCE has demonstrated efficacy in reducing ICP, the impact on cerebral autoregulation and other hemodynamic and metabolic indicators necessitates further investigation. Determining the optimal size of the craniectomy and the timing of CP are also critical factors in patient outcomes. Early CP, performed within three months of controlling increased ICP, appears to be beneficial for overall neurological recovery, as it may reduce the risk of hydrocephalus, facilitate tissue dissection during surgery, and promote neurological function improvement due to the normalization of CSF dynamics and enhancement of CBF. Furthermore, the debate surrounding the necessity of suboccipital CP underscores the need for a more comprehensive understanding of this procedure's role in patient care. As our knowledge of DCE and CP continues to evolve, it is crucial for clinicians to remain informed of the latest evidence and tailor their interventions to optimize patient outcomes in this complex field.

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